U.S. DEPARTMENT OF COMMERCE
Patent and Trademark Office

7-515

SEARCH REQUEST FORM

Requestor's Name: S. Ungar Serial Number: 08/888,202

Date: 7/21/98 Phone: 305-2181 Art Unit: 1642

Search Topic:

Please write a detailed statement of search topic. Describe specifically as possible the subject matter to be searched. Define any terms that may have a special meaning. Give examples or relevant citations, authors keywords, etc., if known. For sequences, please attach a copy of the sequence. You may include a copy of the broadest and/or most relevant claim(s).

Please Search Author Julio C. Pimentel.

+

A method for decreasing fat absorption in mammals by feeding an avian antibody that binds to lipase — Ab is produced in avian eggs. Feeding is oral, in powder form

Thanks - I need this by COB 7/23

Thanks, Susan

1998 JUL 22 AM 8:29

Please rush [illegible]

ore is rush
Substitute for SAF
Dilip

STAFF USE ONLY

Date completed: 7-24-98
Searcher: Bob x8-4291
Terminal time: 73
Elapsed time: prep 40
CPU time:
Total time:
Number of Searches:
Number of Databases: 16

Search Site
___ STIC
✓ CM-1
___ Pre-S

Type of Search
___ N.A. Sequence
___ A.A. Sequence
___ Structure
✓ Bibliographic

Vendors
___ IG Suite
✓ STN
✓ Dialog
___ APS
___ Geninfo
___ SDC
___ DARC/Questel
___ Other

PTO-1590 (9-90)
★ U.S. GPO: 1995-398-798/22489
USCOMM-DC 90-3952

=> fil capl; d que l7; fil biosis; d que l26; fil medl; act ung202medau/a

FILE 'CAPLUS' ENTERED AT 14:07:00 ON 24 JUL 1998
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1967 - 24 Jul 1998 VOL 129 ISS 4
FILE LAST UPDATED: 24 Jul 1998 (980724/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file now supports REG1stRY for direct browsing and searching of all non-structural data from the REGISTRY file. Enter HELP FIRST for more information.

L1 23 SEA FILE=CAPLUS ABB=ON PIMENTEL J?/AU
L2 805 SEA FILE=REGISTRY ABB=ON LIPASE?/CN
L3 19539 SEA FILE=CAPLUS ABB=ON L2
L4 25670 SEA FILE=CAPLUS ABB=ON LIPASE#
L7 0 SEA FILE=CAPLUS ABB=ON L1 AND (L3 OR L4)

FILE 'BIOSIS' ENTERED AT 14:07:00 ON 24 JUL 1998

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 22 July 1998 (980722/ED)
CAS REGISTRY NUMBERS (R) LAST ADDED: 22 July 1998 (980722/UP)

L19 25715 SEA FILE=BIOSIS ABB=ON LIPASE#
L25 92 SEA FILE=BIOSIS ABB=ON PIMENTEL J?/AU
L26 0 SEA FILE=BIOSIS ABB=ON L19 AND L25

FILE 'MEDLINE' ENTERED AT 14:07:01 ON 24 JUL 1998

FILE LAST UPDATED: 23 JUL 1998 (19980723/UP). FILE COVERS 1966 TO DATE.

THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE ANNUAL MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR DETAILS.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

L44 (8351)SEA FILE=MEDLINE ABB=ON LIPASE/CT
L45 (73)SEA FILE=MEDLINE ABB=ON PIMENTEL J?/AU
L46 0 SEA FILE=MEDLINE ABB=ON L45 AND L44

Searched by Barb O'Bryen, STIC 308-4291

```
=> fil capl; d que l11; d que l13; d que l16; s l11 or l13 or l16; fil biosis; d que l43;
fil medl; act ung202med/a

FILE 'CAPLUS' ENTERED AT 14:07:38 ON 24 JUL 1998
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 1998 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is
held by the publishers listed in the PUBLISHER (PB) field (available
for records published or updated in Chemical Abstracts after December
26, 1996), unless otherwise indicated in the original publications.

FILE COVERS 1967 - 24 Jul 1998  VOL 129 ISS 4
FILE LAST UPDATED: 24 Jul 1998  (980724/ED)

  This file contains CAS Registry Numbers for easy and accurate
  substance identification.

  This file now supports REG1stRY for direct browsing and searching
  of all non-structural data from the REGISTRY file.  Enter HELP FIRST
  for more information.


L2          805 SEA FILE=REGISTRY ABB=ON  LIPASE?/CN
L3        19539 SEA FILE=CAPLUS ABB=ON  L2
L4        25670 SEA FILE=CAPLUS ABB=ON  LIPASE#
L5            8 SEA FILE=CAPLUS ABB=ON  ANTI(W)L4
L6       116156 SEA FILE=CAPLUS ABB=ON  ANTIBOD?/OBI
L8          109 SEA FILE=CAPLUS ABB=ON  (L3 OR L4)(L)L6
L10         115 SEA FILE=CAPLUS ABB=ON  L8 OR L5
L11           2 SEA FILE=CAPLUS ABB=ON  L10 AND PHARMAC?/SC


L2          805 SEA FILE=REGISTRY ABB=ON  LIPASE?/CN
L3        19539 SEA FILE=CAPLUS ABB=ON  L2
L4        25670 SEA FILE=CAPLUS ABB=ON  LIPASE#
L5            8 SEA FILE=CAPLUS ABB=ON  ANTI(W)L4
L6       116156 SEA FILE=CAPLUS ABB=ON  ANTIBOD?/OBI
L8          109 SEA FILE=CAPLUS ABB=ON  (L3 OR L4)(L)L6
L10         115 SEA FILE=CAPLUS ABB=ON  L8 OR L5
L12        3208 SEA FILE=CAPLUS ABB=ON  (FAT# OR LIPID#)(3A)ABSOR?
L13           1 SEA FILE=CAPLUS ABB=ON  L10 AND L12


L2          805 SEA FILE=REGISTRY ABB=ON  LIPASE?/CN
L3        19539 SEA FILE=CAPLUS ABB=ON  L2
L4        25670 SEA FILE=CAPLUS ABB=ON  LIPASE#
L5            8 SEA FILE=CAPLUS ABB=ON  ANTI(W)L4
L6       116156 SEA FILE=CAPLUS ABB=ON  ANTIBOD?/OBI
L8          109 SEA FILE=CAPLUS ABB=ON  (L3 OR L4)(L)L6
L10         115 SEA FILE=CAPLUS ABB=ON  L8 OR L5
L14       89101 SEA FILE=CAPLUS ABB=ON  ?CHOLESTEROL?
L15           6 SEA FILE=CAPLUS ABB=ON  L10 AND L14
L16           2 SEA FILE=CAPLUS ABB=ON  RATS/TI AND L15


L47           5 L11 OR L13 OR L16


FILE 'BIOSIS' ENTERED AT 14:07:39 ON 24 JUL 1998
COPYRIGHT (C) 1998 BIOSIS(R)
                    Searched by Barb O'Bryen, STIC  308-4291
```

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 22 July 1998 (980722/ED)
CAS REGISTRY NUMBERS (R) LAST ADDED: 22 July 1998 (980722/UP)

L19 25715 SEA FILE=BIOSIS ABB=ON LIPASE#
L22 298368 SEA FILE=BIOSIS ABB=ON *13006/CC - Metabolism - lipids
L27 414486 SEA FILE=BIOSIS ABB=ON ANTIBOD?
L29 135 SEA FILE=BIOSIS ABB=ON L27(3A)L19
L30 10 SEA FILE=BIOSIS ABB=ON ANTI(W)L19
L31 139 SEA FILE=BIOSIS ABB=ON (L29 OR L30)
L35 39 SEA FILE=BIOSIS ABB=ON L31 AND L22
L43 6 SEA FILE=BIOSIS ABB=ON L35 AND CHYLOMICRON#

FILE 'MEDLINE' ENTERED AT 14:07:39 ON 24 JUL 1998

FILE LAST UPDATED: 23 JUL 1998 (19980723/UP). FILE COVERS 1966 TO DATE.

THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE ANNUAL MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR DETAILS.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

L48 (8351)SEA FILE=MEDLINE ABB=ON LIPASE/CT
L49 (444838)SEA FILE=MEDLINE ABB=ON D24.611.125./CT = antibodies
L50 (80)SEA FILE=MEDLINE ABB=ON L48(L)IM/CT - Subheading IM = immunology
L51 (47)SEA FILE=MEDLINE ABB=ON L50 AND L49
L52 (38767)SEA FILE=MEDLINE ABB=ON CHOLESTEROL(L)BL/CT Subheading BL = blood
L53 2 SEA FILE=MEDLINE ABB=ON L52 AND L51

=> fil wpids; d que 156

FILE 'WPIDS' ENTERED AT 14:11:05 ON 24 JUL 1998
COPYRIGHT (C) 1998 DERWENT INFORMATION LTD

FILE LAST UPDATED: 22 JUL 1998 <19980722/UP>
>>>UPDATE WEEKS:
MOST RECENT DERWENT WEEK 199829 <199829/DW>
DERWENT WEEK FOR CHEMICAL CODING: 199824
DERWENT WEEK FOR POLYMER INDEXING: 199826
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE
>>> D COST AND SET NOTICE DO NOT REFLECT SUBSCRIBER DISCOUNTS - SEE HELP COST FOR DETAILS <<<
>>> DELIMITED FORMAT DALL NOW AVAILABLE <<<

L54 8 SEA FILE=WPIDS ABB=ON PIMENTEL J?/AU
L56 4 SEA FILE=WPIDS ABB=ON ANTIBOD?/TI AND L54

=> d bib ab 156 1-4

L56 ANSWER 1 OF 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 98-312057 [27] WPIDS

DNC C98-096220
TI Increasing efficiency of feed conversion in animals - comprises feeding **antibody** to urease which prevents adverse effects of urease in gut.
DC B04 C03 D13 D16
IN COOK, M E; **PIMENTEL, J**
PA (WISC) WISCONSIN ALUMNI RES FOUND
CYC 72
PI WO 9821979 A1 980528 (9827)* EN 13 pp
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
ADT WO 9821979 A1 WO 97-US16084 970911
PRAI US 96-31403 961119
AB WO 9821979 A UPAB: 980709
Increasing weight gain and/or feed efficiency in an animal comprises feeding a safe amount of an antibody to urease to inhibit the adverse effects of urease. Also claimed is preparation of an agent which prevents the adverse effects of urease produced by organisms in the gut of an animal which comprises injecting hens with jackbean urease, harvesting eggs from the hens and drying the yolks of the eggs or the whole eggs without destroying the antibodies.

The animal is preferably a bird or a mammal. The antibody is present in dried egg yolk or whole egg, and is fed to the animal in its drinking water or another liquid containing the antibody.

USE - The antibodies to urease are used to improve health and are used for treating gastrointestinal diseases, including cancers, which are caused by ammonia. 0.001-1.0 wt.% antibodies are added to the diet or drinking water of an animal.

Dwg.0/0

L56 ANSWER 2 OF 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 98-018228 [02] WPIDS
DNC C98-006773
TI Increasing food conversion in animals - by administering **antibodies** that bind urease particularly egg yolk **antibodies**, especially for improving weight gain in pigs.
DC B04 C03 D13 D16
IN **PIMENTEL, J**
PA (ANIT-N) ANITOX CORP
CYC 75
PI WO 9744060 A1 971127 (9802)* EN 23 pp
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN
US 5741489 A 980421 (9823) 5 pp
AU 9731312 A 971209 (9824)
ADT WO 9744060 A1 WO 97-US8419 970523; US 5741489 A US 96-653604 960524; AU 9731312 A AU 97-31312 970523
FDT AU 9731312 A Based on WO 9744060
PRAI US 96-653604 960524
AB WO 9744060 A UPAB: 980112
Feed conversion efficiency in mammals is increased by including in their diet antibodies (Ab) that bind urease.

The Ab bind jackbean urease and are produced in chicken's eggs. The eggs may be dried and added to the feed, particularly they are freeze-dried then heated at 50-70 deg. C to stabilise Ab.

17-week-old chickens were immunised with 0.2 mg type II-C urease, and the dose repeated 5-6 weeks later, with further doses every 2 months or when the measured Ab titre became low. Ab may be isolated from the eggs by incubating them overnight at 4 deg. C with water, then centrifuging off the aqueous phase and passing through cheesecloth to remove excess fat. The resulting liquid is freeze-dried and heat treated.

USE - Ab are particularly administered to pigs (but are also effective in cows and sheep), particularly at 25-100 mg (as purified Ab)/kg of feed, over at least 4 consecutive weeks. The treatment results in improved weight gain. Ab are already known to protect animals against bacterial infection.

Dwg.0/0

L56 ANSWER 3 OF 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-139459 [14] WPIDS
DNC C96-043794
TI Use of cholecystokinin **antibodies** in avian(s) and humans - useful to elicit a biological response which decreases gastrointestinal motility, reduces satiety and improves feed efficiency..
DC B04 C03 D13 D16
IN COOK, M E; MILLER, C C; **PIMENTEL, J L**
PA (WISC) WISCONSIN ALUMNI RES FOUND
CYC 64
PI WO 9604933 A2 960222 (9614)* EN 22 pp
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UZ VN
AU 9531034 A 960307 (9624)
WO 9604933 A3 960404 (9630)
EP 769964 A1 970502 (9722) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
JP 10504303 W 980428 (9827) 24 pp
ADT WO 9604933 A2 WO 95-US9227 950721; AU 9531034 A AU 95-31034 950721; WO 9604933 A3 WO 95-US9227 950721; EP 769964 A1 EP 95-926766 950721, WO 95-US9227 950721; JP 10504303 W WO 95-US9227 950721, JP 96-507340 950721
FDT AU 9531034 A Based on WO 9604933; EP 769964 A1 Based on WO 9604933; JP 10504303 W Based on WO 9604933
PRAI US 94-286376 940805
AB WO 9604933 A UPAB: 960520
Transferring cholecystokinin (CCK) antibodies (Abs) to an animal in order to increase food efficiency and decrease intestinal motility comprises administering to the animal a CCK Ab contg. substance derived from a producer animal immunised with a CCK peptide. Alternatively, the animal may be immunised with CCK so that the animal produces CCK Abs, which may be passively transferred to the animal's progeny. The methods may also be applied to the transfer of gut peptide Abs, such as bombesin, neuropeptide Y, gastrin or somatostatin Abs.

USE - The Abs may be used to elicit an immune response in avians and mammals in order to increase food efficiency. CCK Abs cause a biological response which decreases gastrointestinal motility, reduces satiety and improves feed efficiency.

ADVANTAGE - By using CCK Abs, individuals in the commercial meat industry can achieve market weight in livestock or poultry using less time and less feed, thereby drastically reducing costs. Use of CCK Abs has many advantages over currently used methods such as admin. of antibiotics (no 'withdrawal time' and no

antibiotics-associated resistance consequences). CCK Abs neutralise CCK but have no known harmful side affects and do not appear to affect meat quality. Cost of using the Abs is low (e.g. CCK Abs may be administered mixed in feed, rather than by individual injections). The ability of CCK Abs to counteract the negative effect of trypsin inhibitor is an advantage. The use of CCK Abs may also be beneficial to humans who are underweight or have problems maintaining their weight. Additionally, individuals with eating disorders would benefit from CCK Abs used because their food intake would be controlled.
Dwg.0/0

L56 ANSWER 4 OF 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 96-129129 [13] WPIDS
DNC C96-040197
TI **Antibodies** against a peptide reverse amino acid sequence esp. choleocystokinin - to mimic the effect of the peptide, e.g. for reducing appetite in birds and mammals.
DC B04 C03 D13
IN COOK, M E; MILLER, C C; **PIMENTEL, J L**
PA (WISC) WISCONSIN ALUMNI RES FOUND
CYC 63
PI WO 9604011 A2 960215 (9613)* EN 22 pp
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG UZ VN
AU 9531035 A 960304 (9623)
WO 9604011 A3 960502 (9630)
ADT WO 9604011 A2 WO 95-US9228 950721; AU 9531035 A AU 95-31035 950721; WO 9604011 A3 WO 95-US9228 950721
FDT AU 9531035 A Based on WO 9604011
PRAI US 94-286113 940805
AB WO 9604011 A UPAB: 960329
The physiological effects of a specific naturally occurring target peptide (A) in an animal are mimicked by admin. of an antibody (Ab) specific to a reverse peptide (RP) having an amino acid sequence the reverse that for (A), from N- to C-termini.
USE - Appetite in mammals (including humans) and birds is suppressed, and gastrointestinal motility increased, by admin. of a reverse choleocystokinin (CCK) Ab. The same effect is achieved by admin. of RP (to generate specific Ab). The method is partic. used in human dietary programmes. (I) are reverse CCK peptides. More generally, RP and reverse antibodies can be used as vaccines in subjects defective in natural prodn. of a peptide or hormone.
ADVANTAGE - Ab are safer than synthetic chemicals currently used to suppress appetite, and their effect is fully reversible.
Dwg.0/0

=> d que 162

L55 3059 SEA FILE=WPIDS ABB=ON LIPASE#
L57 26353 SEA FILE=WPIDS ABB=ON ANTIBOD?
L58 8 SEA FILE=WPIDS ABB=ON ANTI(W)L55
L59 30 SEA FILE=WPIDS ABB=ON L57(3A)L55
L60 36 SEA FILE=WPIDS ABB=ON (L58 OR L59)
L61 37592 SEA FILE=WPIDS ABB=ON FAT# OR LIPID#
L62 4 SEA FILE=WPIDS ABB=ON L60 AND L61

=> d scan 162

L62 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

AN 88-072535 [11] WPIDS
TI New conjugates contg. magnetic material - bound to polyethylene glycol deriv., opt. contg. physiologically active substance.

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

L62 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

AN 90-248416 [33] WPIDS
TI Glyco **lipid** linked, membrane associated protein immunogen - from Eimeria species, useful in vaccines for protecting poultry against coccidiosis.

L62 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

AN 93-047542 [06] WPIDS
TI Analysis of e.g. free fatty acid, cholesterol and lipase - in which measurements are made of at least two items using the same reaction vessel using clinical testing automatic analyser.

L62 4 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

AN 84-158436 [25] WPIDS
TI Kit for assaying hapten in **lipid** contg. sample - esp. testosterone contg. labelled tracer conjugate, solubilising surfactant, opt. **lipase** and 2 **antibodies**.

nothing useful, so doesn't give you Bib/Abs

ALL ANSWERS HAVE BEEN SCANNED

=> dup rem l53,l47,l43

FILE 'MEDLINE' ENTERED AT 14:11:50 ON 24 JUL 1998

FILE 'CAPLUS' ENTERED AT 14:11:50 ON 24 JUL 1998
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 1998 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 14:11:50 ON 24 JUL 1998
COPYRIGHT (C) 1998 BIOSIS(R)
PROCESSING COMPLETED FOR L53
PROCESSING COMPLETED FOR L47
PROCESSING COMPLETED FOR L43
L63 12 DUP REM L53 L47 L43 (1 DUPLICATE REMOVED)

=> d bib ab l63 1-12; fil hom

L63 ANSWER 1 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 97:402436 BIOSIS
DN 99708639
TI Uptake of **chylomicrons** by the liver, but not by the bone marrow, is modulated by lipoprotein lipase activity.
AU Hussain M M; Goldberg I J; Weisgraber K H; Mahley R W; Innerarity T L
CS Gladstone Inst. Cardiovascular Disease, PO Box 419100, San Francisco, CA 94141-9100, USA
SO Arteriosclerosis Thrombosis and Vascular Biology 17 (7). 1997. 1407-1413. ISSN: 1079-5642

LA English
AB We have shown that **chylomicrons** are catabolized by the liver and bone marrow in rabbits and marmosets. In the present investigation, we studied the role of various apolipoproteins and lipoprotein lipase in the clearance of these particles by the liver and bone marrow in rabbits. Incubation of **chylomicrons** with purified apolipoprotein (apo) E or C-II resulted in more rapid clearance of these particles from the plasma, whereas incubation of **chylomicrons** with apoA-I, apoC-I, apoC-III-1, or apoC-III-2 did not affect their clearance rates. Analysis of tissue uptake revealed that the increased plasma clearance rate of **chylomicrons** enriched with apoE or apoC-II was primarily due to enhanced uptake by the liver. The uptake of **chylomicrons** by the bone marrow increased after their enrichment with apoA-I but decreased after their enrichment with apoC-II. Because apoC-II is a cofactor for lipoprotein lipase, we hypothesized that the increased clearance rates were due to faster hydrolysis of **chylomicrons** and rapid generation of **chylomicron** remnants. To test this hypothesis, lipoprotein lipase activity was inhibited by injection of an antilipoprotein **lipase** monoclonal **antibody**. Inhibition of lipoprotein **lipase** retarded clearance of **chylomicrons** from the plasma and decreased their uptake by the liver but did not affect their uptake by the bone marrow. These studies suggest that bone marrow can take up **chylomicrons** in the absence of lipoprotein lipase activity and provide an explanation for the presence of foam cells in the bone marrow of type I hyperlipoproteinemic patients.

L63 ANSWER 2 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:378055 BIOSIS
DN 99100411
TI Chinese hamster ovary cells expressing a cell surface-anchored form of hepatic lipase: Characterization of low density lipoprotein and **chylomicron** remnant uptake and selective uptake of high density lipoprotein-cholesteryl ester.
AU Komaromy M; Azhar S; Cooper A D
CS Res. Inst., PAMF, 860 Bryant St., Palo Alto, CA 94301, USA
SO Journal of Biological Chemistry 271 (28). 1996. 16906-16914. ISSN: 0021-9258
LA English
AB The enzyme hepatic lipase may play several roles in lipoprotein metabolism. Recent investigation has suggested a role for the enzyme in lipoprotein and/or lipoprotein lipid uptake. To study this, a simple isolated system that mimics the in vivo system would be desirable. The enzyme is secreted by the hepatic parenchymal cell but exists, and presumably exerts its effects, while bound to capillary endothelial cells in the liver, adrenal gland, and the ovary. We constructed a cDNA that encodes the expression of a chimeric protein composed of rat hepatic lipase and the signal sequence for the addition of the glycophosphatidylinositol (GPI) anchor from human decay-accelerating factor. When transfected into Chinese hamster ovary (CHO) cells this gave rise to a cell population that had immunoreactive hepatic lipase on the cell surface. Cloning of the transfected cells produced several cell lines that expressed the chimeric protein bound to the cell surface by a GPI anchor. This was documented by demonstrating incorporation of (3H)ethanolamine into anti-hepatic lipase immunoprecipitable material; in addition, hepatic lipase was released from the cells by phosphatidylinositol-specific phospholipase C but not by heparin. Phosphatidylinositol-phospholipase C treatment of cells expressing the anchored lipase released material that comigrated with hepatic lipase on SDS-polyacrylamide gel electrophoresis and was immunoreactive with

antibody to the cross-reacting determinant of GPI anchors. Cell lysates containing the anchored protein contained salt-resistant lipase activity, a known feature of the secreted hepatic lipase; thus it appears that these cells have a surface-anchored hepatic lipase molecule. Although it was not possible to demonstrate lipolysis by the enzyme while it was on the cell surface for technical reasons, the protein produced by these cells was active when studied in cell membranes. The ability of the cells to take up lipoproteins was studied. The cells demonstrated an increased affinity for low density lipoprotein (LDL) receptor mediated uptake of LDL. They did not, however, demonstrate any enhanced binding or removal of **chylomicron** remnants. With respect to LDL and remnants, the cells expressing anchored lipase behaved similarly to CHO cell that expressed secreted hepatic lipase. The cells expressing anchored hepatic lipase had a marked increase in the uptake of high density lipoprotein and high density lipoprotein cholesteryl ester when compared to that seen with CHO cells secreting hepatic lipase. This increase occurred primarily via the selective pathway, and was not reduced by addition of anti-LDL receptor or anti-hepatic **lipase antibodies** or the receptor-associated protein. Together the results suggest that hepatic lipase, when bound to the cell surface by a GPI anchor, plays a role in enhancing lipoprotein uptake. For LDL this may involve the provision of a second foot for particle binding, thus enhancing affinity for the LDL receptor. For **chylomicron** remnants an additional molecule or molecules are necessary to mediate this effect. For HDL, the enzyme facilitates uptake of cholesteryl ester primarily by the selective pathway.

L63 ANSWER 3 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:157671 BIOSIS
DN 98729806
TI Relative roles of the LDL receptor, the LDL receptor-like protein, and hepatic lipase in **chylomicron** remnant removal by the liver.
AU De Faria E; Fong L G; Komaromy M; Cooper A D
CS Res. Inst., Palo Alto Med. Found., Palo Alto, CA 94301, USA
SO Journal of Lipid Research 37 (1). 1996. 197-209. ISSN: 0022-2275
LA English
AB Studies were carried out in mice utilizing inhibitors of several cell surface molecules to evaluate their relative roles in **chylomicron** remnant removal. Anti-LDL receptor antibody inhibited apprx 45% of rapid remnant removal from plasma, prolonged their half life (63 s to 115 s) and reduced hepatic uptake by 45%. Receptor-associated protein (RAP) (1 mg/mouse), a high affinity inhibitor of the LDL receptor-related protein (LRP) and a low affinity inhibitor of the LDL receptor decreased remnant removal apprx 55%, prolonged the half life from 63 s to 230 s, and reduced hepatic uptake by 70%. RAP, but not anti-LDL receptor antibody, inhibited splenic uptake. With both injected together, an incremental effect was seen; plasma removal decreased 60%, T-1/2 increased to 290 s, and hepatic uptake decreased by 80%. Thus, it is likely that virtually all of the very rapid removal of remnants from the plasma by the liver requires the presence of at least one of these members of the LDL receptor family. Anti-hepatic **lipase antibody** caused a small but significant delay in remnant removal from plasma and a larger decrease in hepatic uptake (22.5%). It doubled adrenal uptake. The anti-hepatic **lipase antibody** was not additive with either the anti-LDL receptor antibody or RAP. Anti-rat hepatic **lipase antibody** did not inhibit lipolysis by mouse hepatic lipase, suggesting that lipolysis is not the way hepatic lipase enhances remnant uptake.

Hepatic lipase bound to remnants to a greater degree than it bound to other lipoproteins. Together these data suggest that hepatic lipase may serve as a binding site for **chylomicron** remnants, thereby enhancing their affinity for the liver surface, and thus removal by the proteins of the LDL receptor family. Other molecules may also play a role in removal from the circulation under conditions where the LDL receptor family receptors are absent or occupied.

L63 ANSWER 4 OF 12 CAPLUS COPYRIGHT 1998 ACS
AN 1995:904517 CAPLUS
DN 124:15416
TI A study of reversibly inactivated lipases for use in a morphine-triggered naltrexone delivery system
AU Nakayama, G. R.; ROskos, K. V.; Fritzinger, B. K.; Heller, J.
CS Ixsys, San Diego, CA, USA
SO J. Biomed. Mater. Res. (1995), 29(11), 1389-96
CODEN: JBMRBG; ISSN: 0021-9304
DT Journal
LA English
AB A key component of an implant that can be triggered by external morphine to release naltrexone is an inactivated enzyme that can be activated by morphine and which can then rapidly remove a protective coating surrounding a bioerodible polymer contg. dispersed naltrexone. The authors describe a lipase that was conjugated with O3-carboxymethylmorphine, morphine-.beta.-3-glucuronide and O3-carboxypropylmorphine. The enzyme conjugate was then inactivated by complexation with affinity-purified goat polyclonal antimorphine antibodies. Antibody lipase interactions were measured by pH Stat and ELISA techniques. Affinity consts. of the antibodies detd. by RIA using tritium-labeled morphine were 4.10 .times. 106, 3.18 .times. 106 and 3.38 .times. 107, resp. While a concn. of 10-5M morphine was required to restore lipase activity, it is likely that a combination of correct morphine tether and correct affinity-purified antibody can increase sensitivity to the desired 10-8-10-9M morphine level. Thus, a functioning device can almost certainly be constructed. However, it is unlikely that reactivation times of 1-2 h necessary for clin. usefulness in treatment of narcotic addiction can be achieved.

L63 ANSWER 5 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 94:223103 BIOSIS
DN 97236103
TI Role of hepatic lipase in the uptake and processing of **chylomicron** remnants in rat liver.
AU Shafi S; Brady S E; Bensadoun A; Havel R J
CS Cardiovascular Research, Univ. California, San Francisco, CA 94143-0130, USA
SO Journal of Lipid Research 35 (4). 1994. 709-720. ISSN: 0022-2275
LA English
AB The participation of hepatic lipase in the initial uptake and processing of **chylomicron** remnants was studied in the isolated, perfused rat liver. Hepatic lipase activity was either reduced by preperfusion of livers with heparin or inhibited with specific rat hepatic **lipase antibodies**. (3H)palmitate-labeled **chylomicron** remnants were recirculated through control and treated livers for 15 min; the livers were then flushed, homogenized, and endosome-rich fractions were isolated. Depletion of hepatic lipase activity by both methods reduced the uptake of **chylomicron** remnants and hydrolysis of their component triglycerides by perfused rat livers, but at the same time significantly increased the rate of endocytosis of those **chylomicron** remnants taken up. We conclude that hepatic

lipase facilitates, but is not essential for, the initial uptake of **chylomicron** remnants by rat liver. Furthermore, endocytosis of **chylomicron** remnants does not require binding to hepatic lipase or the associated hydrolysis of remnant lipids.

L63 ANSWER 6 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 95:8409 BIOSIS
DN 98022709
TI An anti-hepatic **lipase antibody** that does not inhibit lipolysis delays removal and reduces hepatic uptake of **chylomicron** remnants in mice.
AU De Faria E C; Komaromy M C; Fong L G; Cooper A D
CS Res. Inst., PAMF, Stanford Univ., Palo Alto, CA, USA
SO 67th Scientific Sessions of the American Heart Association, Dallas, Texas, USA, November 14-17, 1994. Circulation 90 (4 PART 2). 1994. I289. ISSN: 0009-7322
DT Conference
LA English

L63 ANSWER 7 OF 12 CAPLUS COPYRIGHT 1998 ACS
AN 1990:618262 CAPLUS
DN 113:218262
TI **Antibodies** to **lipases** for treatment of acne
IN Miyamoto, Tatsu; Uchida, Ryoichi; Toho, Asami; Ogawa, Tadatake; Tokoro, Toru; Kodama, Yoshikatsu; Yokoyama, Hideaki
PA Kanebo, Ltd., Japan; Gen Corp.
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
PI JP 02121908 A2 900509 Heisei
AI JP 88-274312 881028
DT Patent
LA Japanese
AB A skin lotion for treatment of acne contains antibodies isolated from egg yolk of chickens immunized with lipases produced by Propionibacterium acnes in the cell culture. A lotion consisted of antibodies to P. acnes lipases 0.0002, EtOH 15.0, polyoxyethylene hydrogenated castor oil 1.0, propylene glycol 10.0, and water to 100% by wt.

L63 ANSWER 8 OF 12 CAPLUS COPYRIGHT 1998 ACS
AN 1990:155676 CAPLUS
DN 112:155676
TI Inhibition of hepatic lipase activity impairs chylomicron remnant-removal in **rats**
AU Sultan, Fabrice; Lagrange, Dominique; Jansen, Hans; Griglio, Sabine
CS Inst. Biomed. Cordeliers, Paris, Fr.
SO Biochim. Biophys. Acta (1990), 1042(1), 150-2
CODEN: BBACAQ; ISSN: 0006-3002
DT Journal
LA English
AB [4-14C]cholesteryl oleyl ether-labeled chylomicron remnants were injected into rats which received a specific goat antibody against rat hepatic lipase or a control serum. Chylomicron remnant **cholesterol** ether disappeared from circulation with a higher half-life (2-fold) in antibody-treated rats than in controls. Recovered radioactivity in the liver was 2-fold lower in antibody-treated rats (22.8% vs. 45%). Thus, hepatic lipase may strongly promote chylomicron remnant **cholesterol** ether uptake by the liver.

L63 ANSWER 9 OF 12 CAPLUS COPYRIGHT 1998 ACS
AN 1987:115538 CAPLUS

DN 106:115538
TI Assay of human pancreatic lipase in biological fluids using a non-competitive enzyme immunoassay
AU Carrere, Jaqueline; Galabert, Claude; Thouvenot, Jean Paul; Figarella, Catherine
CS Lab. Biochim., Hop. Purpan, Toulouse, 13009, Fr.
SO Clin. Chim. Acta (1986), 161(2), 209-19
CODEN: CCATAR; ISSN: 0009-8981
DT Journal
LA English
AB A sandwich enzyme immunoassay has been developed for human pancreatic lipase (I) using polystyrene balls coated with specific IgG as the 1st antibody and peroxidase-labeled IgG as the 2 antibody. The detection limit was 0.5 .mu.g/L. Good parallelism was obsd. with the curves obtained from std. I and from I present in blood serum, pancreatic juice, and duodenal contents, demonstrating that the assay may be used to measure the level of I in different biol. fluids. Mean values of I in human sera were 12.3 .mu.g/L in adults and 4.5 in newborns. In all cases, a good correlation was found in serum between the catalytic activity and the enzyme immunoassay. I was detectable in amniotic fluid at the 18th wk of pregnancy, but at a very low level (0.95 .mu.g/L). In pancreatic juice, I concn. was 14.6% of the total protein content. A study on cystic fibrosis patients showed a poor correlation between blood I concn. and fat malabsorption, thus underlying the difficulty in assessing pancreatic function by the measurement of serum pancreatic enzymes. The use of the I assay in duodenal contents should permit better assessment of pancreatic function in patients presenting a severe or borderline defect in **fat** digestion and **absorption**.

L63 ANSWER 10 OF 12 MEDLINE DUPLICATE 1
AN 81255951 MEDLINE
DN 81255951
TI Accumulation of intermediate density lipoprotein in plasma after intravenous administration of hepatic triglyceride lipase antibody in rats.
AU Murase T; Itakura H
SO ATHEROSCLEROSIS, (1981 Jun) 39 (3) 293-300.
Journal code: 95X. ISSN: 0021-9150.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198111
AB In an attempt to define the role of hepatic triglyceride lipase in plasma lipoprotein metabolism, in vivo experiments using an antibody specifically prepared against this enzyme were conducted in rats. The antibody gamma globulins were injected into rats three times during a 40 min period. Control rats received non-immune rabbit gamma globulins prepared in the same way as the immune gamma globulins. After treatment, blood was taken and the plasma was separated. Plasma lipoproteins were fractionated by ultracentrifugation into VLDL, IDL, LDL and HDL. Treatment of recipient rats with the antibody significantly increased cholesterol, phospholipid and protein concentrations in the IDL fraction. These concentrations were also elevated in the LDL fraction. However, we speculate that this increase represents the accumulation of small remnants rather than bona fide LDL. VLDL compositions in antibody-treated rats did not differ from those in control animals. In HDL, only the phospholipid level was elevated in antibody-treated rats. The data of the present study indicate that

hepatic triglyceride lipase mediates the catabolism of remnant lipoproteins by the liver.

L63 ANSWER 11 OF 12 MEDLINE
AN 80004177 MEDLINE
DN 80004177
TI Hepatic endothelial lipase antiserum influences rat plasma low and high density lipoproteins in vivo.
AU Kuusi T; Kinnunen P K; Nikkila E A
SO FEBS LETTERS, (1979 Aug 15) 104 (2) 384-8.
Journal code: EUH. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198001

L63 ANSWER 12 OF 12 BIOSIS COPYRIGHT 1998 BIOSIS
AN 78:151888 BIOSIS
DN BA65:38888
TI **ANTIBODIES** TO LIPO PROTEIN **LIPASE** APPLICATION TO PERFUSED HEART.
AU SCHOTZ M C; TWU J-S; PEDERSEN M E; CHEN C-H; GARFINKEL A S; BORENSZTAJN J
CS RES. VETERANS ADM., WADSWORTH HOSP. CENT., LOS ANGELES, CALIF. 90073, USA.
SO BIOCHIM BIOPHYS ACTA 489 (2). 1977 214-224. CODEN: BBACAQ ISSN: 0006-3002
LA English
AB An antibody was prepared against purified rat heart lipoprotein **lipase**. This **antibody** showed marked species specificity. It inhibited almost totally the lipoprotein lipase activity from all rat tissues examined (i.e., heart, adipose, postheparin plasma and mammary gland), while having no effect on the activity of lipoprotein lipase partially purified from rabbit, guinea pig and bovine heart and from bovine milk. The antibody also had no effect on the hepatic lipase activity of rat postheparin plasma. After antibody to rat heart lipoprotein lipase was recirculated for 5 min through isolated rat hearts, little or no lipoprotein lipase activity could be detected in the perfusate during 0-20 s of a subsequent non-recirculating perfusion with buffer containing 1 unit heparin/ml. Following recirculation of **antibody** to lipoprotein **lipase** for 10 min and a non-recirculating perfusion with buffer for 2 min, the hearts no longer oxidized any significant amounts of 14C-labeled palmitate **chylomicron** triacylglycerol fatty acid to 14CO2 during a 15 min perfusion. The data give compelling evidence that the functional fraction of lipoprotein lipase in hearts is at the endothelial cell surface accessible to lipoprotein **lipase antibody**.

FILE 'HOME' ENTERED AT 14:12:04 ON 24 JUL 1998

```
? show files

File  10:AGRICOLA 70-1998/Jul
         (c) format only 1998 The Dialog Corporation
File  60:CRIS/USDA 1998/Jun
         (c) format only 1998 The Dialog Corporaion pl
File 203:AGRIS 1974-1998/Jul
         Dist by NAL, Intl Copr. All rights reserved
File  50:CAB Abstracts 1972-1998/Jun
         (c) 1998 CAB International
File  76:Life Sciences Collection 1982-1998/Jun
         (c) 1998 Cambridge Sci Abs
File  73:EMBASE 1974-1998/Jul W4
         (c) 1998 Elsevier Science B.V.
File 144:Pascal 1973-1998/Jun
         (c) 1998 INIST/CNRS
File 357:Derwent Biotechnology Abs 1982-1998/Aug B2
         (c) 1998 Derwent Publ Ltd
File 358:Current BioTech Abs 1983-1998/Aug
          Royal Soc Chem & DECHEMA
File 315:ChemEng & Biotec Abs 1970-1998/Jul
         (c)1998 RoySocChm,DECHEMA,FizChemie
File  74:Int.Pharm.Abs. 1970-1998/Jul
         (c) 1998 Amer.Soc.of Health-System Pharm.
? ds


Set     Items   Description
S1        142   AU=((PIMENTEL, J?) OR (PIMENTEL J?))
S2      45982   LIPASE?
S3        297   ANTIBOD?(3N)S2 OR ANTI(W)S2
S4          0   S1 AND S2   author
S5     234937   EGG OR EGGS
S6          2   S3(5N)S5
S8     663210   ORAL?
S10         1   S3 AND S8
S12    670424   FAT OR FATS OR LIPID OR LIPIDS
S13        65   S3 AND S12
S14    177414   S12(5N)(ABSOR? OR METABOL?)
S15        18   S3 AND S14

?

? rd s6

>>>Duplicate detection is not supported for File 60.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
     S18       1  RD S6 (unique items)
? t s18/7


 18/7/1     (Item 1 from file: 73)
DIALOG(R)File  73:EMBASE
(c) 1998 Elsevier Science B.V. All rts. reserv.

10044615   EMBASE No: 96239077
  Development and evaluation of an ELISA method for the determination of
lipoprotein lipase mass concentration comparison with a commercial,
one-step enzyme immunoassay
  Antikainen M.; Suurinkeroinen L.; Jauhiainen M.; Ehnholm C.; Taskinen
```

M.-R.
National Public Health Institute, Department of Biochemistry, Mannerheimintie 166, FIN-00300 Helsinki Finland
European Journal of Clinical Chemistry and Clinical Biochemistry (Germany) , 1996, 34/7 (547-553)
CODEN: EJCBE ISSN: 0939-4974
LANGUAGES: English SUMMARY LANGUAGES: English
We developed a non-competitive, enzyme-linked, immunosorbent assay (ELISA) for the quantitation of lipoprotein lipase (LPL) in human postheparin plasma using affinity-purified antihuman milk lipoprotein ****lipase**** ****antibodies**** produced in chicken ****eggs**** and a monoclonal antibody directed against human lipoprotein lipase. We compared our ELISA method with a commercially available sandwich-enzyme immunoassay (Markit-F LPL EIA Kit, Dainippon Pharmaceutical Co, Ltd. Osaka, Japan). The reference values for lipoprotein lipase catalytic activity concentration and mass concentration in healthy Finns were determined. Lipoprotein lipase activity concentration (mean plus or minus SD) was 297 plus or minus 112 U/l in women, and mass concentration as measured by the ELISA method was 1058 plus or minus 367 microg/l. The corresponding values for men were 247 plus or minus 97 U/l and 815 plus or minus 207 microg/l, respectively. Across the whole concentration range of the ELISA method, the control samples' intra- and inter-assay coefficients of variation (CV) were 5.1% and 6.5%, respectively. The correlation between the ELISA and EIA methods was good, r = +0.81. The importance of the correct standardisation of immunoassays is discussed.
?

? rd s10

>>>Duplicate detection is not supported for File 60.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
S19 1 RD S10 (unique items)
? t s19/7

19/7/1 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

9905461 EMBASE No: 96078932
The evaluation of the effects of an association of dimeticone and a polyenzymatic preparation (Zymoplex (R)) on the colic fermentations induced by lactulose in healthy volunteers
EVALUATION DE L'EFFET D'UNE ASSOCIATION DE DIMETICONE ET D'UNE PREPARATION POLYENZYMATIQUE (ZYMOPLEX (R)) SUR LES FERMENTATIONS COLIQUES INDUITES PAR LE LACTULOSE CHEZ LE VOLONTAIRE SAIN
Moreau J.; Fondarai J.; Frexinos J.
Serv. Gastro-Enterologie/Nutrition, C.H.U. Rangeuil, 31054 Toulouse France
Medecine et Chirurgie Digestives (France) , 1996, 25/1 (43-47)
CODEN: MCDGB ISSN: 0047-6412
LANGUAGES: French
?

? rd s15

>>>Duplicate detection is not supported for File 60.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records

S20 16 RD S15 (unique items)
? t s20/7/1-16

20/7/1 (Item 1 from file: 50)
DIALOG(R)File 50:CAB Abstracts

02483161 CAB Accession Number: 911438932
Plasma lipoprotein ****metabolism**** in lean and in ****fat**** chickens produced by divergent selection for plasma very low density lipoprotein concentration.
Griffin, H.; Acamovic, F.; Guo, K.; Peddie, J.
Agricultural and Food Research Council's Institute for Grassland and Animal Production, Poultry Department, Roslin, Midlothian EH25 9PS, UK.
Journal of Lipid Research vol. 30 (8): p.1243-1250
Publication Year: 1989
ISSN: 0022-2275
Language: English
Document Type: Journal article
Plasma lipoprotein metabolism was studied in lines of chickens produced by selection for high and low plasma very-low-density lipoprotein (VLDL) concentration. Rates of VLDL secretion were measured by estimating the rate of accumulation of triacylglycerol (TG) in plasma after intravenous injection of anti-lipoprotein ****lipase**** ****antibody****. Clearance of VLDL-TG and its uptake into liver and adipose tissue was examined using radioactively labelled VLDL synthesized in vivo. The rate of VLDL secretion was about threefold higher in the high VLDL line than that in the leaner, low VLDL-line (6.7 vs. 2.1 micro mol VLDL TG/h ml plasma). Clearance of VLDL from the circulation of the low VLDL line was much faster than that of the high VLDL line (half-time of 3.7 and 13.6 min, respectively). The proportion of administered radiolabel taken up by the abdominal fat pad was substantially greater in the fat line than in the lean line (11.9 vs. 4.8%, respectively). Lipoprotein lipase activities in leg muscle and heart were consistently greater in the low-VLDL line and beta -hydroxybutyrate concentrations in the plasma of the low-VLDL line were significantly greater than those in the high-VLDL line (0.86 vs. 0.48 micro mol/ml). The results show that the approximately ten-fold difference in plasma VLDL concentration between lines is primarily due to markedly different rates of hepatic VLDL production and that selection has made a major effect on partitioning of VLDL triglyceride between adipose and other tissues. It is proposed that a direction of fatty acids to oxidation rather than VLDL synthesis in the liver of birds of the low-VLDL line is a major cause of their low rate of VLDL secretion and makes an important contribution to improved efficiency of protein utilization. In addition, preferential use of VLDL-TG and beta -hydroxybutyrate may reduce amino acid oxidation by muscle. 27 ref.

20/7/2 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10694554 EMBASE No: 98114800
Identification of the epitope of a monoclonal antibody that inhibits heparin binding of lipoprotein lipase: New evidence for a carboxyl-terminal heparin-binding domain
Sendak R.A.; Melford K.; Kao A.; Bensadoun A.
A. Bensadoun, Div. of Nutritional/Biological Sci., Cornell University, Ithaca, NY 14853 United States
Journal of Lipid Research (United States) , 1998, 39/3 (633-646)
CODEN: JLPRA ISSN: 0022-2275

Searched by Barb O'Bryen, STIC 308-4291

DOCUMENT TYPE: Journal Article
LANGUAGES: ENGLISH SUMMARY LANGUAGES: ENGLISH
NUMBER OF REFERENCES: 41

A panel of 13 monoclonal ****antibodies**** to avian lipoprotein ****lipase**** (LPL) was screened for inhibition of LPL binding to primary avian adipocytes. One monoclonal antibody, designated xCAL (monoclonal antibody to chicken adipose lipoprotein lipase) 3-6a, was found to inhibit the binding of LPL to primary avian adipocytes. In solid phase assays, xCAL 3-6a inhibited the binding of LPL to both heparan sulfate and heparin. XCAL 3-6a did not inhibit the catalytic activity of the avian enzyme. The monoclonal antibody was not found to cross-react significantly with bovine lipoprotein lipase. In order to determine the location of the epitope of xCAL 3-6a on lipoprotein lipase, several avian lipoprotein lipase deletion mutants were constructed and produced as glutathione S-transferase (GST) fusion proteins in E. coli. These mutants were screened for their ability to react with xCAL 3-6a using Western blotting. The minimum continuous fragment of lipoprotein lipase that was required for reactivity contained the amino acids 310 to 450. Site-directed mutagenesis of basic residues 321,405, 407, 409, 415, and 416 revealed that Arg 405 is necessary for the interaction of LPL with xCAL 3-6a. Additional deletions of either the amino-or carboxyl-terminal portion of the fragment containing residues 310-450 resulted in loss of antibody binding, suggesting that the epitope is a discontinuous one that is formed when the termini are brought together through protein folding. Heparin-Sepharose chromatography of wild-type LPL and a mutant LPL in which the well-characterized heparin- binding sequence (Arg 281-Lys 282-Arg 284) has been mutated was carried out in the presence and absence of xCAL 3-6a. These experiments indicate that lipoprotein lipase contains a heparin-binding domain, in addition to Arg 281- Arg 284, that can be blocked by xCAL 3-6a.

20/7/3 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

10431329 EMBASE No: 97236376
Uptake of chylomicrons by the liver, but not by the bone marrow, is modulated by lipoprotein lipase activity
Hussain M.M.; Goldberg I.J.; Weisgraber K.H.; Mahley R.W.; Innerarity T.L.
Dr. T.L. Innerarity, GICD, PO Box 419100, San Francisco, CA 94141-9100 USA
Arteriosclerosis, Thrombosis, and Vascular Biology (USA) , 1997, 17/7 (1407-1413)
CODEN: ATVBF ISSN: 1079-5642
DOCUMENT TYPE: Journal
LANGUAGES: English SUMMARY LANGUAGES: English
NUMBER OF REFERENCES: 48

We have shown that chylomicrons are catabolized by the liver and bone marrow in rabbits and marmosets. In the present investigation, we studied the role of various apolipoproteins and lipoprotein lipase in the clearance of these particles by the liver and bone marrow in rabbits. Incubation of chylomicrons with purified apolipoprotein (apo) E or C-II resulted in more rapid clearance of these particles from the plasma, whereas incubation of chylomicrons with apoA-I, apoC-I, apoC-III1, or apoC-III2 did not affect their clearance rates. Analysis of tissue uptake revealed that the increased plasma clearance rate of chylomicrons enriched with apoE or apoC-II was primarily due to enhanced uptake by the liver. The uptake of chylomicrons by the bone marrow increased after their enrichment with apoA-I but decreased after their enrichment with apoC-II. Because apoC-II is a cofactor for lipoprotein lipase, we hypothesized that the increased clearance rates were due to faster hydrolysis of chylomicrons and rapid

generation of chylomicron remnants. To test this hypothesis, lipoprotein lipase activity was inhibited by injection of an anti-lipoprotein ****lipase**** monoclonal ****antibody****. Inhibition of lipoprotein ****lipase**** retarded clearance of chylomicrons from the plasma and decreased their uptake by the liver but did not affect their uptake by the bone marrow. These studies suggest that bone marrow can take up chylomicrons in the absence of lipoprotein lipase activity and provide an explanation for the presence of foam cells in the bone marrow of type I hyperlipoproteinemic patients.

20/7/4 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

10035080 EMBASE No: 96216996
Chinese hamster ovary cells expressing a cell surface-anchored form of hepatic lipase: Characterization of low density lipoprotein and chylomicron remnant uptake and selective uptake of high density lipoprotein-cholesteryl ester

Komaromy M.; Azhar S.; Cooper A.D.
Research Institute, PAMF, 860 Bryant St., Palo Alto, CA 94301 USA
Journal of Biological Chemistry (USA) , 1996, 271/28 (16906-16914)
CODEN: JBCHA ISSN: 0021-9258
LANGUAGES: English SUMMARY LANGUAGES: English

The enzyme hepatic lipase may play several roles in lipoprotein metabolism. Recent investigation has suggested a role for the enzyme in lipoprotein and/or lipoprotein lipid uptake. To study this, a simple isolated system that mimics the in vivo system would be desirable. The enzyme is secreted by the hepatic parenchymal cell but exists, and presumably exerts its effects, while bound to capillary endothelial cells in the liver, adrenal gland, and the ovary. We constructed a cDNA that encodes the expression of a chimeric protein composed of rat hepatic lipase and the signal sequence for the addition of the glycophosphatidylinositol (GPI) anchor from human decay- accelerating factor. When transfected into Chinese hamster ovary (CHO) cells this gave rise to a cell population that had immunoreactive hepatic lipase on the cell surface. Cloning of the transfected cells produced several cell lines that expressed the chimeric protein bound to the cell surface by a GPI anchor. This was documented by demonstrating incorporation of (3H)ethanolamine into anti-hepatic lipase immunoprecipitable material; in addition, hepatic lipase was released from the cells by phosphatidylinositol- specific phospholipase C but not by heparin. Phosphatidylinositol- phospholipase C treatment of cells expressing the anchored lipase released material that comigrated with hepatic lipase on SDS-polyacrylamide gel electrophoresis and was immunoreactive with antibody to the cross-reacting determinant of GPI anchors. Cell lysates containing the anchored protein contained salt-resistant lipase activity, a known feature of the secreted hepatic lipase; thus it appears that these cells have a surface-anchored hepatic lipase molecule. Although it was not possible to demonstrate lipolysis by the enzyme while it was on the cell surface for technical reasons, the protein produced by these cells was active when studied in cell membranes. The ability of the cells to take up lipoproteins was studied. The cells demonstrated an increased affinity for low density lipoprotein (LDL) receptor mediated uptake of LDL. They did not, however, demonstrate any enhanced binding or removal of chylomicron remnants. With respect to LDL and remnants, the cells expressing anchored lipase behaved similarly to CHO cell that expressed secreted hepatic lipase. The cells expressing anchored hepatic lipase had a marked increase in the uptake of high density lipoprotein and high density lipoprotein cholesteryl ester when compared to that seen with CHO cells secreting hepatic lipase. This increase occurred primarily via the selective pathway, and was not reduced by addition of

anti-LDL receptor or anti-hepatic ****lipase**** ****antibodies**** or the receptor-associated protein. Together the results suggest that hepatic lipase, when bound to the cell surface by a GPI anchor, plays a role in enhancing lipoprotein uptake. For LDL this may involve the provision of a second foot for particle binding, thus enhancing affinity for the LDL receptor. For chylomicron remnants an additional molecule or molecules are necessary to mediate this effect. For HDL, the enzyme facilitates uptake of cholesteryl ester primarily by the selective pathway.

20/7/5 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

9865097 EMBASE No: 96041159
Coenzyme A-independent monoacylglycerol acyltransferase from rat intestinal mucosa
Tsujita T.; Miyazaki T.; Tabei R.; Okuda H.
Department of Medical Biochemistry, School of Medicine, Ehime University, Shigenobu, Onsen-gun, Ehime 791-02 Japan
Journal of Biological Chemistry (USA) , 1996, 271/4 (2156-2161)
CODEN: JBCHA ISSN: 0021-9258
LANGUAGES: English SUMMARY LANGUAGES: English
Rat intestinal mucosa contains high diacylglycerol-synthesizing activity (monoacylglycerol acyltransferase (MGAT) activity) due to monoacylglycerol and fatty acid, independently of coenzyme A and ATP. MGAT activity was purified from rat intestinal mucosa by successive chromatography separations on DEAE-cellulose, CM- Sephadex, and anti-IgG-Sepharose against rat pancreatic lipase. The enzyme was electrophoretically homogeneous, and its molecular weight was 49,000, which is identical with that of rat pancreatic ****lipase****. Immunoblotting analysis with ****antibody**** against rat pancreatic ****lipase**** showed one immunoreactive protein with an estimated molecular weight of 49,000. The activity of the purified enzyme was completely inhibited by addition of the antibody. Using immunocytochemical techniques, it was found that immunoreactive protein against rat pancreatic lipase was uniformly distributed within the absorptive cells of the intestine but was absent from the microvillar membrane. The MGAT activity of intestinal mucosal homogenate was inhibited by about 65% by addition of ****antibody**** against rat pancreatic ****lipase**** . Trioleoylglycerol- and dioleoylglycerol-hydrolyzing activities of the purified enzyme and pancreatic lipase were inhibited by addition of intestinal mucosa extract. These results suggest that pancreatic lipase is present in intestinal absorptive cells and that it may contribute to resynthesis of diacylglycerol from monoacylglycerol and fatty acids in these cells.

20/7/6 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

9346778 EMBASE No: 94285227
Establishment of enzyme-linked immunosorbent assays for lipoprotein ****lipase**** with newly developed ****antibodies****
Kawamura M.; Gotoda T.; Mori N.; Shimano H.; Kozaki K.; Harada K.; Shimada M.; Inaba T.; Watanabe Y.; Yazaki Y.; Yamada N.
Third Dept. of Internal Medicine, Faculty of Medicine, University of Tokyo, Hongo 7-3-1, Tokyo 113 Japan
J. LIPID RES. (USA) , 1994, 35/9 (1688-1697)
CODEN: JLPRA ISSN: 0022-2275
LANGUAGES: English SUMMARY LANGUAGES: English
We developed eight new ****antibodies**** against lipoprotein

****lipase**** (LPL), which included polyclonal antibodies raised against recombinant human LPL produced by transformant cells and two synthetic peptides corresponding to either amino (N)- or carboxy (C)-terminus of human LPL. With these antibodies, we established three effective sandwich enzyme-linked immunosorbent assays (ELISAs) for LPL, which enabled us to examine LPL mass not only in the postheparin plasma from human, rat, mouse, and guinea pig but also in the media and lysates of cultured cells. All of the developed antibodies showed high affinities for LPL, but their binding to LPL did not always influence the lipolytic activity of the enzyme. Interestingly, although the anti-C- terminus antibody should bind to a common epitope of human and mouse LPL, its binding selectively suppressed only human LPL activity. Because amino acid sequence surrounding the epitope is common to both LPLs, difference in the sequence outside the epitope will contribute to the selective suppression of LPL activity by the antibody. Our results also suggested that both termini of LPL would be exposed on the surface of the molecule because they were fully accessible to antibodies and that the N-terminus of LPL would be functionally less important because binding of the anti-N-terminus antibody did not affect human LPL activity. The ELISAs were further utilized to demonstrate the presence of C-terminus truncated LPL protein in the postheparin plasma of an LPL-deficient patient, to map an epitope of the anti-C-terminus antibody within residues 433-436, and to gain insight into the structure-function relationship of the LPL molecule. Availability of effective antibodies that have different epitope specificities and different inhibitory effects on LPL function will be of great use in immunological analysis of LPL.

20/7/7 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

6378003 EMBASE No: 87114662
****Antibody**** against human lipoprotein ****lipase****
Etienne J.; Noe L.; Millot F.; et al.
Department of Internal Medicine, Centre Hospitalo-Universitaire St. Antoine-Tenon, Paris FRANCE
ATHEROSCLEROSIS (IRELAND) , 1987, 64/2-3 (201-209)
CODEN: ATHSB
LANGUAGES: ENGLISH
A polyclonal ****antibody**** against human lipoprotein ****lipase**** (LPL) was prepared. LPL from post-heparin plasma was first purified by heparin Sepharose 4B affinity chromatography. Protein impurities co-eluted with LPL were then eliminated by electrophoresis in the presence of ampholytes. Antithrombin III was identified in this fraction of protein impurities by immunodiffusion against a human antithrombin antiserum, while no antithrombin III could be detected in the purified LPL fraction. Immunodiffusion revealed a single line of precipitation between this antibody and human post-heparin plasma LPL. When pre-incubated with a constant activity of highly purified post-heparin plasma LPL (2.7 mU/75 mul), an equal volume of the anti-LPL antiserum, either pure or diluted to 1/32 caused complete inhibition of the enzyme activity. Half maximal inhibition was observed at a dilution of approximately 1/200. By using a secondary antibody, it was shown that antiserum inhibited LPL activity by means of its immunoglobulins. This antibody was able to inhibit LPL from human adipose tissue, indicating that human LPL released from endothelial cell membranes has common antigenic determinants with adipose tissue LPL.

20/7/8 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

6173101 EMBASE No: 86168161
Hormone-sensitive lipase from bovine adipose tissue
Cordle S.R.; Colbran R.J.; Yeaman S.J.
Department of Biochemistry, University of Newcastle upon Tyne, Newcastle upon Tyne, NE1 7RU UNITED KINGDOM
BIOCHIM. BIOPHYS. ACTA (NETHERLANDS) , 1986, 887/1 (51-57)
CODEN: BAMRD
SERIES: SER. MOL. CELL RES.
LANGUAGES: ENGLISH
Hormone-sensitive lipase has been purified to near homogeneity from bovine perirenal adipose tissue. The purification method involves isoelectric precipitation at pH 5.0, followed by partial solubilisation in Triton N-101 and ion-exchange chromatography on DE-52. After additional solubilisation, the enzyme is further purified by chromatography on phenyl-Sepharose and heparin-Sepharose. This procedure can be completed within three working days and yields approx. 30 units of enzyme with a specific activity of 30 U/mg. The enzyme has been identified as a polypeptide of M(r) 84,000 by affinity labelling with (sup 3H)diisopropyl fluorophosphate. This polypeptide comprises approx. 60-80% of the protein in the final preparation, as judged by scanning densitometry of SDS-polyacrylamide gels stained with silver or with Coomassie blue R. The polypeptide of M(r) 84,000 serves as a substrate for cyclic AMP-dependent protein kinase, phosphorylation correlating with activation of the ****lipase****. Polyclonal ****antibody**** to the ****lipase**** has been raised in a rabbit and shown to specifically cross-react with the M(r) 84,000 subunit.

20/7/9 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

5902116 EMBASE No: 85147626
Antibody against rat adipose tissue lipoprotein lipase
Etienne J.; Noe L.; Rossignol M.; et al.
Departments of Biochemistry and Medicine, Centre Hospitalo-Universitaire Saint Antoine Tenon FRANCE
BIOCHIM. BIOPHYS. ACTA (NETHERLANDS) , 1985, 834/1 (95-102)
CODEN: BBLLA
SERIES: SER. LIPIDS LIPID METAB.
LANGUAGES: ENGLISH
To facilitate detailed studies of rat adipose tissue lipoprotein lipase regulation, a high titre polyclonal antibody was raised against purified rat adipose tissue lipoprotein lipase (in a goat). The first stage of the purification of the lipoprotein lipase was carried out with heparin-Sepharose affinity chromatography. In the second stage we took advantage of the binding property of lipoprotein lipase to ampholytes. These ampholytes, used during this second step, do not have to be eliminated prior to injecting the enzyme preparation into the animal. They have neither toxic nor antigenic effects on the animal; moreover, their presence does not affect the antigenic potency of the lipoprotein lipase. When pre-incubated with a constant amount of adipose tissue lipoprotein lipase (8 mU/75 mul), an equal volume of the antiserum raised either pure or diluted up to 1/50 resulted in complete inhibition of enzyme activity, and half maximal inhibition was observed at a dilution of 1/800. The antibody was effective in inhibiting rat heart lipoprotein lipase but not salt-resistance hepatic lipase. Immunodiffusion revealed a single line of precipitation between this antibody and the adipose tissue lipoprotein lipase.

20/7/10 (Item 9 from file: 73)

DIALOG(R)File 73:EMBASE

5798636 EMBASE No: 85044146
Plasma apoprotein changes after selective inhibition of hepatic triglyceride lipase in rat
Grosser K.J.; Schrecker O.; Greten H.
Klinisches Institut fur Herzinfarktforschung an der Medizinischen Universitatsklinik, D-6900 Heidelberg GERMANY, WEST
ATHEROSCLEROSIS (IRELAND) , 1984, 53/3 (233-239)
CODEN: ATHSB
LANGUAGES: ENGLISH
Selective inhibition of hepatic triglyceride ****lipase**** (HTGL) by specific ****antibodies**** in rats led to an altered VLDL apoprotein composition. Apoprotein analysis by isoelectric focusing revealed a new protein band in VLDL and an increase in apoprotein E (apo E) content. Apoproteins in LDL and HDL remained unchanged. Electronmicroscopy showed a significant increase in particle size of VLDL from 452 to 497 Angstrom with no significant changes in LDL and HDL diameters.

20/7/11 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

2371075 EMBASE No: 81143531
Accumulation of intermediate density lipoprotein in plasma after intravenous administration of hepatic triglyceride ****lipase**** ****antibody**** in rats
Murase T.; Itakura H.
Dept. Int. Med., Univ. Tokyo JAPAN
ATHEROSCLEROSIS (IRELAND) , 1981, 39/3 (293-300)
CODEN: ATHSB
LANGUAGES: ENGLISH
In an attempt to define the role of hepatic triglyceride lipase in plasma lipoprotein metabolism, in vivo experiments using an antibody specifically prepared against this enzyme were conducted in rats. The antibody gamma globulins were injected into rats three times during a 40 min period. Control rats received non-immune rabbit gamma globulins prepared in the same way as the immune gamma globulins. After treatment, blood was taken and the plasma was separated. Plasma lipoproteins were fractionated by ultracentrifugation into VLDL, IDL, LDL and HDL. Treatment of recipient rats with the antibody significantly increased cholesterol, phospholipid and protein concentrations in the IDL fraction. These concentrations were also elevated in the lDL fraction. However, we speculate that this increase represents the accumulation of small remnants rather than bona fide LDL. VLDL compositions in antibody-treated rats did not differ from those in control animals. In HDL, only the phospholipid level was elevated in antibody-treated rats. The data of the present study indicate that hepatic triglyceride lipase mediates the catabolism of remnant lipoproteins by the liver.

20/7/12 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

2346083 EMBASE No: 81118254
Acid lipase cross-reacting material in Wolman disease and cholesterol ester storage disease
Burton B.K.; Reed S.P.
Dept. Ped., Bowman Gray Sch. Med., Winston-Salem, N.C. 27103 USA

AM. J. HUM. GENET. (USA) , 1981, 33/2 (203-208)
CODEN: AJHGA
LANGUAGES: ENGLISH
Material cross-reacting with ****antibodies**** to acid ****lipase**** was demonstrated in fibroblasts of three patients with Wolman disease and three with cholesterol ester storage disease. Quantitation of the immunologically cross-reacting material (CRM) by a single radial immunodiffusion method revealed normal levels in both mutant cell types. CRM specific activity toward triolein and cholesteryl oleate was reduced about 200-fold in the Wolman disease fibroblasts and 50- to 100-fold in the cholesterol ester storage disease cells when compared to normal.

20/7/13 (Item 12 from file: 73)
DIALOG(R)File 73:EMBASE

2254815 EMBASE No: 81025891
Purification of an anti-lipoprotein lipase antiserum
Dosne A.M.; Etienne J.; Noe L.; et al.
CHU St-Antoine, Paris FRANCE
PAROI ARTERIELLE (FRANCE) , 1980, 6/1 (49-53)
CODEN: PARTD
LANGUAGES: ENGLISH SUMMARY LANGUAGES: FRENCH
Lipoprotein lipase is an enzyme difficult to isolate in pure form and, until now, the antisera prepared against it have not been monospecific. The present experiments show how a crude antiserum prepared with bovine milk lipoprotein lipase, can be made more specific through suitable adsorption. The crude antiserum was prepared by injecting milk lipoprotein lipase prepared by heparin Sepharose affinity chromatography. Immunodiffusion techniques indicated that the antiserum contained antibodies to proteins other than lipoprotein lipase (bovine milk and serum proteins) and that these antibodies could be eliminated by adsorption with bovine serum and a beta-casein preparation.

20/7/14 (Item 13 from file: 73)
DIALOG(R)File 73:EMBASE

1255761 EMBASE No: 79023315
A regulatory study in rat heart of heparin-releasable lipoprotein lipase
Kronquist K.E.; Pedersen M.E.; Schotz M.C.
VA Wadsworth Hosp. Cent., Los Angeles, Calif. USA
CIRCULATION (USA) , 1978, 58/4II (No. 50)
CODEN: CIRCA
LANGUAGES: ENGLISH

20/7/15 (Item 14 from file: 73)
DIALOG(R)File 73:EMBASE

028822 EMBASE No: 74029238
Studies on hormone sensitive lipase and lipoprotein lipase in adipose tissue
Okuda H.; Fujii S.
Dept. Enzyme Physiol., Inst. Enzyme Res., Sch. Med., Tokushima Univ., Tokushima JAPAN
J.BIOCHEM. (TOKYO) (JAPAN) , 1973, 73/6 (1195-1203)
CODEN: JOBIA
LANGUAGES: ENGLISH

Rat liver esterase (EC 3.1.1.1) was purified to yield an electrophoretically homogeneous component. Anti liver esterase antibody was prepared using this purified preparation. The ****antibody**** effectively precipitated the ****lipase**** (EC 3.1.1.3) and esterase activities of adipose lipase fraction, and the esterase activity of the adipose tissue esterase fraction. Based on these and previous results, it is suggested that adipose tissue lipase is composed of esterase and lipid. Anti liver esterase antibody also precipitated both hormone sensitive lipase and lipoprotein lipase of adipose tissue. The lipolytic activity of hormone sensitive lipase was maximum at pH 6.8 in the absence of serum and at pH 8.0 in the presence of serum. The activity was markedly activated by serum, which stimulated the formation of an enzyme substrate complex. In the presence of 1M NaCl, serum did not activate lipase activity and the maximum activity remained at pH 6.8. The lipoprotein lipase activity was clearly inactivated by acetone treatment. From these results, it is suggested that hormone sensitive lipase and lipoprotein lipase may be the same entity.

20/7/16 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

13190023 PASCAL No.: 97-0453966
Uptake of chylomicrons by the liver, but not by the bone marrow, is modulated by lipoprotein lipase activity
MAHMOOD HUSSAIN M; GOLDBERG I J; WEISGRABER K H; MAHLEY R W; INNERARITY T L
Gladstone Institute of Cardiovascular Disease University of California, San Francisco, United States; Cardiovascular Research Institute, University of California, San Francisco, United States; Department of Medicine, College of Physicians & Surgeons of Columbia University, New York, NY, United States; Department of Pathology, University of California, San Francisco, United States; Department of Medicine, University of California, San Francisco, United States
Journal: Arteriosclerosis, thrombosis, and vascular biology, 1997, 17 (7) 1407-1413
ISSN: 1079-5642 Availability: INIST-19104; 354000067991350290
No. of Refs.: 48 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: United States
Language: English
We have shown that chylomicrons are catabolized by the liver and bone marrow in rabbits and marmosets. In the present investigation, we studied the role of various apolipoproteins and lipoprotein lipase in the clearance of these particles by the liver and bone marrow in rabbits. Incubation of chylomicrons with purified apolipoprotein (apo) E or C-II resulted in more rapid clearance of these particles from the plasma, whereas incubation of chylomicrons with apoA-I, apoC-I, apoC-III,, or apoC-III SUB 2 did not affect their clearance rates. Analysis of tissue uptake revealed that the increased plasma clearance rate of chylomicrons enriched with apoE or apoC-II was primarily due to enhanced uptake by the liver. The uptake of chylomicrons by the bone marrow increased after their enrichment with apoA-I but decreased after their enrichment with apoC-Il. Because apoC-II is a cofactor for lipoprotein lipase, we hypothesized that the increased clearance rates were due to faster hydrolysis of chylomicrons and rapid generation of chylomicron remnants. To test this hypothesis, lipoprotein lipase activity was inhibited by injection of an antilipoprotein ****lipase**** monoclonal ****antibody****. Inhibition of lipoprotein ****lipase**** retarded clearance of chylomicrons from the plasma and decreased their uptake by the liver but did not affect their uptake by the bone marrow. These studies suggest that bone marrow can take up chylomicrons in the absence of lipoprotein lipase activity and provide an

explanation for the presence of foam cells in the bone marrow of type I hyperlipoproteinemic patients.

?

? logoff hold

24jul98 13:29:36 User219845 Session D517.2